Case Report

Haemolytic–uraemic syndrome in an adult male with Aeromonas hydrophila enterocolitis

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Introduction

Verotoxin-producing Escherichia coli is the major causative agent of the haemolytic–uraemic syndrome in adults [1,2]. Shigella is the other organism that may also lead to typical post-diarrhoea haemolytic–uraemic syndrome [3]. In 1991, a case of haemolytic–uraemic syndrome triggered by Aeromonas hydrophila was reported in a 23-month-old female infant [4], but thus far this association had not been found in adults. We observed a case of haemolytic–uraemic syndrome associated with A. hydrophila in a previously healthy adult male.

Case

A previously healthy sailor aged 36 years old was admitted because of severe uraemia. Two months before admission, he complained of watery diarrhoea associated with intermittent abdominal cramping pain after eating seafood. Nausea and vomiting was followed by bloody diarrhoea in the 14 days before admission. He had only received symptomatic treatment. Three days before admission, he developed oliguria and progressive dyspnea.

On admission, blood pressure was 160/100 mmHg and his temperature was 39.2°C. He was very pale and oedematos without evidence of purpura or hepatosplenomegaly. Chest X-ray examination revealed pulmonary oedema. Haemoglobin was 3.5 g/dl, platelet count 24 000/mm³ and white blood count 11 100/mm³ with 97% neutrophils. Lactate dehydrogenase (LDH) was 4200 U/l. Peripheral blood smear showed anisocytosis, poikilocytosis, target cells, rouleaux, dacrocytes and fragmented red blood cells, pointing to the presence of microangiopathic anaemia.

Serum urea was 293 mg/dl, creatinine 35.1 mg/dl, sodium 129 mmol/l, potassium 3.9 mmol/l, albumin 2.8 g/dl, calcium 7.2 mg/dl, phosphate 6.3 mg/dl, C₃ 77.5 mg/dl, C₄ 9.7 mg/dl, ANA negative. Arterial blood gas analysis showed pH 7.28, pCO₂ 17.5 mmHg, pO₂ 91.5 mmHg, HCO₃ 8.2 mmol/l; oxygen saturation was 96.3%.

Radioimmunoassay of hepatitis B antigen, anti-hepatitis C virus antibody and VDRL showed negative results. The Coombs’ test was negative. Prothrombin time and partial thromboplastin time were normal. Renal ultrasonography showed borderline size kidneys with somewhat irregular contours, increased cortical echogenicity, reduced cortical mass and no dilatation of the pelvicalyceal systems. Urinalysis showed proteinuria (500 mg/dl), haematuria (50 RBC/high power field) and sterile leukocyturia (70 WBC/high power field) with many granular casts (seven casts/lower power field). He was treated with antihypertensive drugs, packed red cell transfusions and cefetazidine. Emergency haemodialysis was arranged for severe uraemia and fluid overload. A fluctuating course of aphasia and delirium of unknown cause was seen at the end of the first week after admission. Fever and diarrhoea subsided and no significant haemolysis was noted at the end of the third week after admission. However, anuria and azotaemia persisted throughout the period of admission.

A percutaneous renal biopsy was performed at the end of the second week. The majority of the glomeruli were sclerosed, with only few glomeruli showing segmental or lobular preservation. Fragmentation of red cells was seen in these areas (Figure 1). There was also severe interstitial fibrosis associated with tubular atrophy. Mild intimal thickening was seen in a few small vessels (Figure 2). An immunofluorescence study showed deposits of fibrinogen, C₃ and C₄ in the partially sclerosed glomeruli. Regular haemodialysis three times a week was arranged for end-stage renal disease.

Blood and stool samples for culture were obtained immediately after admission and processed by routine
infections. No predisposing factor was identified in our patient, the only relevant history being his recent intake of some seafood which may have contained *Aeromonas* producing haemolysin and cytotoxin [5,6].

Bogdanovic et al. [4] proposed that the pathogenesis of haemolytic–uraemic syndrome caused by *A. hydrophila* and that caused by verotoxin-producing *E. coli* infection is similar.

Many strains of *A. hydrophila* can be isolated from the faeces of patients with diarrhoea [7], and all strains produce Vero cell cytotoxin and haemolysin [7,8]. *Aeromonas hydrophila* produces a cytotoxin which damages the membranes of various types of cells [9,10]. This toxin may also damage the endothelial cells of the kidney.

Antibiotics do not improve the course of enterohaemorrhagic *E. coli* infection in haemolytic–uraemic syndrome. Although no controlled trials have validated antibiotic therapy for diarrhoea induced by *Aeromonas*, clinical improvement has occurred with the third generation cephalosporin ceftriaxone. Our patient with severe bacteraemia was treated successfully with ceftriaxone. Whether antibiotics will generally improve the course of *A. hydrophila* infection and the haemolytic–uraemic syndrome has to be determined.

The present case illustrates that *A. hydrophila* can cause post-diarrhoeal haemolytic–uraemic syndrome, and that contaminated water or food is the source of infection.

**References**


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